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PEER VICTIMIZATION AND THE DEVELOPMENT OF ANXIETY AND
DEPRESSIVE SYMPTOMS: THE ROLES OF STRESS PHYSIOLOGY AND
GENDER

A Thesis Presented

by

Leigh Ann Holterman

to

The Faculty of the Graduate College

of

The University of Vermont

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For the Degree of Master of Arts
Specializing in Psychological Science

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Abstract

The overall goal of the current study was to determine whether experiences of relational and physical victimization were related to anxiety and depressive symptoms in a sample of emerging adults. This study also investigated whether these associations were moderated by gender, as well as by sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) reactivity to peer stress. Although work in this area has focused on children (e.g., Cullerton-Sen & Crick, 2005; Rudolph et al., 2009), it appears the presence and function of victimization changes with age, and the negative effects of victimization can last through early adulthood (e.g., Gros et al., 2010; Kumpulainen et al., 1999; Roth et al., 2002). Despite the potential for victimization to influence outcomes in emerging adults, research on these associations is lacking in this age group (Heilbron & Prinstein, 2008). A goal of the current study was to examine these processes in an older sample. Additionally, as individuals may react to peer victimization differently, factors that may help explain these differences were investigated. Specifically, evidence suggests that the interaction of the SNS and the PNS may serve as a moderator in the relationship between stressors and adjustment outcomes (Cummings et al., 2007; El-Sheikh et al., 2009; Obradović et al., 2010). Further, research suggests that different patterns of interaction of the SNS and the PNS provide important information in the prediction of adjustment outcomes (El-Sheikh et al., 2009) and that both systems must be examined in order to more fully understand the relationship between physiological reactivity and adjustment outcomes (Beauchaine, 2001). Thus, in the current study, the interaction between two physiological measures, SNS reactivity to stress (as measured by skin conductance reactivity [SCL-R]) and PNS reactivity to stress (as measured by respiratory sinus arrhythmia [RSA-R]), was examined as a moderator of the association between peer victimization and adjustment outcomes. The moderating role of gender was also examined.

Two hundred and forty-six emerging adults participated in the current study (74% female; $M_{age} = 18.77$) and were recruited from introductory psychology courses at a northeastern public university. Participants' SCL-R and RSA-R were assessed using a stress protocol during which they discussed an experience of relational victimization (e.g., being left out). Levels of relational and physical victimization, anxiety and depressive symptoms, and gender were gathered using self-report.

Findings suggested that both physical and relational victimization were related to both anxiety and depressive symptoms. Additionally, females were more likely to experience relational victimization than males, while males were more likely to experience physical victimization than females. Relational victimization was related to depressive symptoms only in individuals demonstrating *coactivation* (i.e., blunted RSA withdrawal and increased SCL-R) and *coinhibition* (i.e., RSA withdrawal and blunted SCL-R) patterns of stress reactivity, although the interaction for this effect only approached conventional levels of statistical significance. These patterns may have emerged as a result of the breakdown of regulation in the physiological response to stress, with either the SNS or the PNS failing to perform adequately (El-Sheikh & Erath, 2011; El-Sheikh et al., 2009). These findings suggest that experiences of victimization are related to negative adjustment outcomes in emerging adults, as well as highlight potential areas that may serve as mechanisms for future interventions.

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Introduction

Peer victimization, or receiving aggressive behavior from peers, is a common phenomenon in the lives of youth and its consequences can vary in magnitude and quality. Victimization occurs when a child is repeatedly exposed to negative actions perpetrated with the intent to cause harm by at least one peer (Grills & Ollendick, 2002; Olweus, 1997). Researchers have struggled to understand the mechanisms through which children are affected by negative behaviors at the hands of their peers. Complicating matters is the fact that victimization can occur in various manifestations, such as overt (e.g., physical violence) or relational (e.g., gossiping, manipulation and peer exclusion) (Olweus, 1997; Rudolph, Troop-Gordon, & Flynn, 2009; Sijtsema, Shoulberg, & Murray-Close, 2011), and researchers have documented gender differences in both the forms and outcomes of victimization experiences (Crick & Bigbee, 1998; Paquette & Underwood, 1999; Vuijk, van Lier, Crijnen, & Huizink, 2007). Additionally, although increasing evidence suggests that individual differences in the physiological stress response play a role in the relationship between adversity, such as peer victimization, and adjustment outcomes (Rudolph, Troop-Gordon, & Granger, 2010; Scarpa & Ollendick, 2003), research in this area is still in its nascent stages. Further, the interaction of multiple stress systems, such as the sympathetic (SNS) and parasympathetic (PNS) nervous systems, must be examined in order to more fully understand the implications of patterns of physiological reactivity for adjustment outcomes (Beauchaine, 2001). Thus, the overall goal of the current study was to determine whether experiences of relational and physical victimization were related to anxiety and depressive symptoms in a sample of emerging

adults. This study also investigated whether these associations were moderated by interactions between sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) reactivity to peer stress. Finally, given potential gender differences in the stress response (e.g., Taylor, 2006), the moderating role of gender in these associations was assessed.

All types of peer victimization have been associated with numerous negative outcomes for youth, potentially leading to decreased emotional and physical well-being. The focus of prior research that has linked peer victimization experiences with anxiety and depressive symptoms has been on children ranging from preschool-age through adolescence (e.g., Boivin, Hymel, & Bukowski, 1995; Crick & Bigbee, 1998; Crick, Casas, & Ku, 1999; Crick & Grotpeter, 1995; Crick & Nelson, 2002; Gros, Gros, & Simms, 2010; Hodges & Perry, 1999; Klomek, Marrocco, Kleinman, Schonfeld, & Gould, 2008; Prinstein, Boergers, & Vernberg, 2001; Rudolph et al., 2009; Troop-Gordon & Ladd, 2005; Vuijk et al., 2007). The limited research exploring the correlates of peer victimization during emerging adulthood is notable, particularly because preliminary research suggests that the negative effects of victimization can last through adolescence and early adulthood (Gros et al., 2010; Kumpulainen, Räsänen, & Henttonen, 1999; Roth, Coles, & Heimberg, 2002).

Although most of what is known about the associations between peer victimization and adjustment outcomes is based on research in younger samples, a discussion of these processes is warranted as they may occur in emerging adulthood as well. Victimized children may develop problems with self-control, internalizing

problems, or may behave in submissive ways (Crick & Bigbee, 1998). These maladaptive behaviors could both aggravate difficulties with peers and lead to future issues with mental health (Crick & Bigbee, 1998; Crick & Nelson, 2002; Hawker & Boulton, 2000; Hodges, Malone, & Perry, 1997). In fact, children identified by their peers as victims tended to report more feelings of distress than those who were not (Rudolph, Troop-Gordon, & Granger, 2011).

Internalizing symptoms, including symptoms of depression, anxiety, withdrawal, and loneliness, have been repeatedly linked to social difficulties, including peer victimization, for youth (Ginsburg, La Greca, & Silverman, 1998; La Greca & Lopez, 1998; Verduin & Kendall, 2008). Some researchers have argued that internalizing pathology may serve as a risk factor for maltreatment by peers. For instance, children with internalizing symptoms (e.g. social anxiety, withdrawn behavior) tend to demonstrate social skills deficits, such as displaying poor social problem-solving skills and attaining fewer social goals (Stewart & Rubin, 1995), anticipating more negative outcomes and using less constructive interactions in role-playing tasks (Morgan & Banerjee, 2006), and displaying less social competence and positive affect during interactions with peers (Panella & Henggeler, 1986).

Although it is likely that youth with internalizing pathology may become targets of aggression by peers as a result of these social skills deficits, the relationship between victimization and internalizing symptoms is likely reciprocal such that peer victimization may also serve as a potent risk factor for the development of internalizing symptoms (Reijntjes, Kamphuis, Prinzie, & Telch, 2010; Verduin & Kendall, 2008). Peer

difficulties may result in internalizing adjustment problems for youth in a variety of ways. Children may evaluate themselves negatively and feel that they do not fit in with their peers as a result of difficult social interactions (Crick & Bigbee, 1998; Crick & Dodge, 1994; Vuijk et al., 2007). In turn, these children may develop internalizing problems as a result of these negative self-evaluations (Crick & Bigbee, 1998; Vuijk et al., 2007). Victimized children may also resist involvement in social activities, effectively preventing themselves from gaining normative social experiences and the exposure required for developing social skills. This resulting social isolation may place them at risk for negative outcomes such as feelings of distress, depression, and trait and social anxiety, in both the short and long term (Hanish & Guerra, 2002; Roth et al., 2002).

In fact, mounting evidence suggests that being rejected or excluded by others in the social group may lead to the experience of anxiety, and may in fact be the most common cause (Baumeister & Leary, 1995; Baumeister & Tice, 1990). Peer harassment has been linked repeatedly with increased symptoms of depression and anxiety in children and early adolescents (Boivin et al., 1995; Grills & Ollendick, 2002; Nishina, Juvonen, & Witkow, 2005; Paul & Cillessen, 2003; Slee, 1994). Additionally, victims of aggression were more likely to report anxiety symptoms than non-victims in childhood and early adolescence (Hawker & Boulton, 2000), and children who reported higher levels of peer victimization also reported more symptoms of anxiety and depression (Craig, 1998; Grills & Ollendick, 2002; Slee, 1995). On the other hand, peer acceptance has been shown to be positively related to psychological adjustment during childhood and early adolescence (Oberle, Schonert-Reichl, & Thomson, 2010)

Some longitudinal research findings lend support for the proposition that victimized youth develop internalizing problems over time. For instance, a longitudinal study by Hanish and Guerra (2002) found evidence that, consistent across gender and age in elementary school children, peer victimization was related to a number of negative outcomes, including symptoms of anxiety and depression. Findings indicated that early victimization predicted later adjustment problems (i.e., aggression, anxiety/depression) above and beyond the effects of current victimization and prior adjustment problems. However, the effects depended on the type of analyses conducted (person-centered versus variable-centered; Hanish & Guerra, 2002). Overall, despite some mixed evidence, based on research with child and adolescent samples there is both theoretical and empirical reason to expect that victimization serves as a risk factor for anxiety and depression; thus, the theoretical orientation of the present study was in reference to victimization predicting these internalizing problems.

Victimization in Emerging Adulthood

As is evident above, much of the prior research on the relationships between peer victimization and anxiety and depressive symptoms has been conducted on children (Crick et al., 1999; Crick & Nelson, 2002; Hodges & Perry, 1999; Prinstein et al., 2001; Vuijk et al., 2007). Little work to date has examined these associations and processes in samples of emerging adults. Emerging adulthood is an important developmental period to consider when assessing the relation between peer victimization and symptoms of anxiety and depression for several reasons. Emerging adulthood encompasses the late teens and early twenties, particularly ages 18-25. This period is considered to be distinct

empirically and in theory from both adolescence and young adulthood (Arnett, 2000). According to Arnett (as cited in Munsey, 2006), there are five defining features of this stage of life, such that it is the age of identity exploration, instability, self-focus, feeling in-between, and possibilities. Youth in this age group are beginning to experience increased independence, increased importance of romantic relationships, and the challenges of peer cohabitation and conflict resolution (Gros et al., 2010). This is a time of greater instability during which individuals are more reliant on their peers for support, having left their families and often not yet married (Arnett, 2000). Experiences of peer victimization in the face of these challenges of development may undermine emerging adults' sense of support from peers, thereby putting them at risk for symptoms of anxiety and depression.

However, research on the associations between peer victimization and adjustment outcomes, including symptoms of anxiety and depression, in emerging adulthood is lacking (Heilbron & Prinstein, 2008). Some research does suggest that the negative effects of victimization can last through adolescence and early adulthood; for instance, being teased in youth was related to internalizing symptoms such as trait anxiety, social anxiety, and depression in adulthood (Gros et al., 2010; Kumpulainen et al., 1999; Roth et al., 2002). Though this evidence does suggest that peer victimization is related to symptoms of anxiety and depression in emerging adulthood, relatively little work has investigated the concurrent relationships between these constructs during this developmental period. Therefore, an aim of the current study was to extend previous research regarding associations between peer victimization and symptoms of anxiety and

depression beyond childhood and adolescence to examine whether these processes are similar in a developmental period that is relatively understudied.

Physical and Relational Forms of Victimization

Traditionally, research on peer victimization focused primarily on the overt forms, such as being the target of physical violence and intimidation, teasing, threats, and verbal attacks (Crick & Bigbee, 1998; Crick & Nelson, 2002; Cullerton-Sen & Crick, 2005). However, the assessment of only overt forms of victimization overlooks those who experience instances of relational victimization, such as being the target of gossip, social manipulation, and behaviors intended to harm via damage to relationships (Crick & Bigbee, 1998; Crick, Grotpeter, & Bigbee, 2002; Cullerton-Sen & Crick, 2005). In recent years, some studies have included a global measure of victimization (i.e., including relational and physical victimization experiences; Boivin et al., 1995; Hodges & Perry, 1999; Troop-Gordon & Ladd, 2005); however, this approach prevents the exploration of mechanisms linking the different types of victimization and internalizing problems. As such, it is important for researchers to examine the different outcomes associated with the various types of victimization.

Within the theories developed with child and adolescent studies, there are several potential mechanisms linking relational victimization with internalizing pathology. For instance, children who experience relational victimization may fail to develop sufficient emotion regulation and coping skills (Rudolph et al., 2009), fail to develop close, supportive friendships, and may experience an erosion of trust in their peers (Crick, Casas, & Nelson, 2002; Vuijk et al., 2007). Relational victimization may produce

feelings of social alienation in children, leading to adjustment problems such as internalizing symptoms (Gros, Gros, & Simms, 2010; Troop-Gordon & Ladd, 2005). In contrast, the unique associations between physical victimization (i.e., controlling for relational victimization) and anxiety/depressive symptoms are less clear in the literature. For instance, one study suggests that, while relational victimization predicted internalizing symptoms (for males), physical victimization did not (in either gender; Cullerton-Sen & Crick, 2005). However, since both forms may undermine feelings of trust in peers, as well as produce feelings of alienation (Troop-Gordon & Ladd, 2005), it is possible that both physical and relational forms of victimization serve as risk factors for internalizing problems such as symptoms of anxiety and depression as a result of similar mechanisms.

A growing body of research indicates both relational and physical victimization are associated with negative internalizing outcomes, such as loneliness and avoidance/anxiety, in children (Crick & Bigbee, 1998; Crick & Grotpeter, 1995), as well as anxiety for early and late adolescents, although, in this case, the relationship for physical victimization was moderated by gender (Yeung Thompson & Leadbeater, 2013). Both forms of victimization have also been concurrently and prospectively linked with depressive symptoms, across different age groups (Boivin et al., 1995; Hodges, Boivin, Vitaro, & Bukowski, 1999; Klomek et al., 2008; Prinstein et al., 2001; Yeung Thompson & Leadbeater, 2013). Taken together, then, research indicates that both forms of victimization may be related to symptoms of depression and anxiety. To provide a broader and more complete assessment of victimization, the present study examined the

unique associations between relational and physical forms of victimization and symptoms of anxiety and depression.

An important characteristic of this study is that it extended findings of these relationships in childhood and adolescence to the relatively understudied age group of emerging adulthood. To date, the vast majority of research on the association between these two forms of victimization and symptoms of anxiety and depression has been conducted with children and adolescents. As research on these associations in emerging adults is sparse, it is important to explore whether similar associations are evident in this developmental period. There are several reasons to expect that relational forms of peer victimization may be a particularly potent risk factor for internalizing problems in emerging adulthood. As children get older and enter adolescence, physical victimization may become less adaptive; thus, its frequency may decrease over time (Hawley, 1999, 2003). Further, physical victimization carries greater risk of serious injury and thus, legal risk, as youth get older; therefore, relational forms may represent a safer alternative method of expressing anger and frustration in the peer group (Cairns, Cairns, Neckerman, Ferguson, & Gariépy, 1989; Prinstein et al., 2001). In addition, relational aggression, as a more subtle form, tends to become more accepted during adolescence (Heilbron & Prinstein, 2008). Thus, experiences of relational victimization may increase with age (Heilbron & Prinstein, 2008), peak in early adolescence, and then exhibit an overall decline (Orpinas, McNicholas, & Nahapetyan, 2014). However, as relationships tend to be given great importance during adolescence and emerging adulthood, relational forms of aggression and victimization may be particularly salient during this time (Arnett,

2000; Parker, Rubin, Erath, Wojslawowicz, & Buskirk, 2006; Prinstein et al., 2001).

Given the state of the literature, there is reason to believe that relational victimization may be more strongly associated with anxiety and depressive symptoms than physical victimization among emerging adults. This suggests that it is important to investigate the relative influence of relational versus physical victimization on symptoms of anxiety and depression in older samples, when relational victimization is more normative and social relationships are highly salient. Therefore, the first goal of the current study was to investigate the unique associations between both physical and relational victimization and symptoms of anxiety and depression in a sample of emerging adults, and explore whether differences emerged between the two forms.

The Role of Gender in Victimization

Gender may also play an important role in individuals' experiences of physical and relational victimization. In fact, the study of relational victimization began in an effort to gain a more gender-balanced understanding of the role of peer victimization in the lives of youth by assessing forms of victimization hypothesized to be more common among girls (Crick, Casas, & Nelson, 2002). Females may face more relational victimization than males because females' social goals tend to be centered on maintaining strong relationships, prioritizing dyadic relationships, and highly valuing social evaluation (Dempsey & Storch, 2008; Paquette & Underwood, 1999; Rose & Rudolph, 2006; Rudolph et al., 2009). Thus, females may be more likely to be targeted by behaviors that undermine these goals, such as relational aggression. On the other hand, males may face more physical victimization from peers than females because

interpersonal situations that tend to be stressful and distressing for males are those that involve physical dominance, peer status, competition, and instrumental concerns (Crick, Grotpeter, & Bigbee, 2002; Gabriel & Gardner, 1999; Maccoby, 1990; Rudolph, 2002; Shih, Eberhart, Hammen, & Brennan, 2006). Their social goals tend to be agentic in nature and oriented toward social and reputational status, even when these goals require acting in a socially undesirable fashion (Caravita & Cillessen, 2012; LaFontana & Cillessen, 2010; Rose & Rudolph, 2006; Rudolph et al., 2009). Therefore, males may be more likely to face behaviors from peers that target these goals, like physical victimization.

Consistent with these hypotheses, in some studies conducted with child and adolescent samples, the inclusion of relational victimization in research has resulted in the discovery of more victimized girls than previously found (Crick & Bigbee, 1998). This work suggests that males and females likely experience victimization at a similar frequency, but the qualitative nature of the experience typically varies by gender. In fact, evidence from several studies indicates that females tend to face significantly more relational victimization than males, whereas males experience significantly more physical victimization than females (Crick & Bigbee, 1998; Crick & Nelson, 2002; Cullerton-Sen & Crick, 2005; Vuijk et al., 2007). However, research regarding whether girls experience more relational victimization than boys has been mixed, with findings of gender differences dependent on the chosen methodology, age group, and informant (Crick, Casas, & Nelson, 2002; Prinstein et al., 2001; Rose & Rudolph, 2006). Additionally, while research on these gender differences in emerging adulthood is sparse, it is possible

that gender differences may continue into older developmental periods (Rose & Rudolph, 2006). Given these mixed findings, the second goal of this study was to examine whether there were gender differences in the frequency of the experiences of physical and relational victimization during emerging adulthood.

Gender may also play a key role in understanding the relationship between victimization and anxiety and depressive symptoms (Vuijk et al., 2007). Even with the same level of relational and physical victimization, males and females may respond to these peer experiences differently. For instance, relational victimization may be more likely to lead to negative outcomes, such as internalizing problems, for females because they effectively harm the relationship-oriented social goals typical in female peer groups (Crick & Bigbee, 1998). This is perhaps why relational victimization has been shown to predict social and psychological adjustment problems for females and not males in several studies of children, adolescents, and emerging adults (Crick & Nelson, 2002; Dempsey & Storch, 2008; Paquette & Underwood, 1999; Rudolph, 2002). However, findings of gender differences in outcomes associated with relational victimization are mixed (Troop-Gordon & Ladd, 2005). For instance, Crick and Nelson (2002) reported that relational victimization was related to loneliness, distress, internalizing problems, and externalizing problems for both boys and girls. In other research on adolescents and emerging adults, relational victimization was prospectively related to symptoms of social phobia for both genders (Dempsey & Storch, 2008; Storch, Masia-Warner, Crisp, & Klein, 2005). In one study on children, relational victimization was related to internalizing symptoms for males but not females (Cullerton-Sen & Crick, 2005). Despite

this, mounting evidence indicates that females appear to respond with more distress and internalizing outcomes in response to relational victimization and interpersonal stressors such as gossip or disagreements with friends than males do across the developmental periods of childhood, adolescence, and emerging adulthood (Crick et al., 2002b; Dempsey & Storch, 2008; Leadbeater, Kuperminc, Blatt, & Hertzog, 1999; Prinstein et al., 2001; Rudolph & Hammen, 1999; Rudolph et al., 2000).

Alternatively, the status- and dominance-oriented nature of males' social goals may put them at greater risk for negative outcomes such as internalizing pathology when they face physical victimization, as these experiences may directly target their highly-valued social goals. In fact, some evidence suggests that overt victimization is a significant predictor of depressive symptoms for just male adolescents (Prinstein et al., 2001). However, other work has demonstrated associations between physical victimization and depressive symptoms across both genders in childhood (Storch, Nock, Masia-Warner, & Barlas, 2003). Given these mixed findings, the third goal of the current study was to examine whether gender moderated the unique associations between relational and physical victimization and anxiety and depressive symptoms in a sample of emerging adults. Based on the evidence above using samples from childhood through emerging adulthood, I expected that relational victimization would be more strongly associated with symptoms of anxiety and depression among females whereas physical victimization would be more strongly associated with these symptoms among males.

Autonomic Reactivity

Increasing evidence suggests that individual differences in the physiological stress response play a role in the relationship between adversity, such as peer victimization, and adjustment outcomes (Rudolph et al., 2010; Scarpa & Ollendick, 2003). The human stress response system demonstrates a significant amount of variability across individuals both in baseline levels, and in how it reacts to external stimuli, which can lead to a vast array of outcomes (Del Giudice, Ellis, & Shirtcliff, 2011).

The human stress response is made up of two different systems. The first is a slow-acting mechanism that operates through the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis primes the body for exposure to stress through the release of glucocorticoids (e.g. salivary cortisol), which in turn suppress systems that are not immediately necessary and increase available energy (Obradović, 2012). Chronically high or low levels of glucocorticoids have been shown to affect adaptation in human health (for a review of the HPA system, see Gunnar & Quevedo, 2007), leading to outcomes such as anxiety and depressive symptoms (Chrousos & Gold, 1992).

The second human stress system is the quick-acting autonomic nervous system (ANS), which is the focus of the current study. The ANS is the system in the adrenal medulla pathway responsible for releasing catecholamines into the bloodstream in order to quickly initiate the fight-or-flight response (Chrousos & Gold, 1992; Obradović, 2012; Rudolph et al., 2010). The ANS is made up of two main branches, the sympathetic nervous system (SNS; responsible for initiating physiological arousal) and the parasympathetic nervous system (PNS; the activation of which is responsible for

restoring homeostasis and decreasing arousal; Obradović, 2012; Sijtsma et al., 2011).

Typically, in the face of a stressor, the SNS becomes activated, while the PNS withdraws, leading to an increased physiological response known as fight-or-flight (Sijtsma et al., 2011).

SNS activity in the face of a stressor can be examined through measures of electrodermal activity, such as skin conductance reactivity (SCL-R; Dawson, Schell, & Filion, 2007; Murray-Close, 2013). Increased sweat production, which is an indicator of increased SNS activity, results in an increase in the electrical conductance of the outer layer of the skin (see Dawson et al., 2007, for a detailed explanation). This can then be measured with electrodes by passing an electrical current through the skin to measure the conductivity (Murray-Close, 2013). SNS reactivity has been hypothesized to be related to the behavioral inhibition system (BIS), the behavioral activation system (BAS), as well as emotional experiences such as anger and excitement (Murray-Close, 2013).

PNS functioning in response to a stressor can be measured by assessing respiratory sinus arrhythmia reactivity (RSA-R). The vagus nerve, or the tenth cranial nerve, is responsible for providing the body with efferent pathways that help humans rapidly interact with stressors in their environments, either to mobilize or calm down (Porges, 2007). Upon exhalation, vagal input is increased, leading to an increased regulatory influence of the PNS on the heart and a subsequent deceleration in heart rate. Upon inhalation, the opposite effect occurs (Porges, 2007). Respiratory sinus arrhythmia (RSA), also referred to as vagal tone, is the measure of this variability in heart rate that occurs naturally with the respiratory cycle (Murray-Close, 2013). Thus, baseline RSA

reflects increases and decreases in heart rate during respiration. RSA can change in the face of a stressor, which is known as RSA reactivity (RSA-R). RSA-R is considered to be a good measure of the PNS response to stress, as PNS functioning can influence heart rate variability (Berntson, Cacioppo, & Quigley, 1991; Murray-Close, 2013).

RSA-R is hypothesized to influence a variety of adjustment outcomes.

Theoretically, greater RSA withdrawal in response to a stressor is hypothesized to reflect strong emotion regulation capabilities and has been linked with lower levels of internalizing symptoms (Graziano & Derefinko, 2013). Additionally a failure to exhibit RSA withdrawal during a stressor may lead to coping difficulties, and may place youth at risk for depressive symptoms (Hinnant & El-Sheikh, 2009).

Important to the current study, the reactivity level of the ANS to stress may serve as a powerful moderator of the associations between environmental risk and adjustment. One framework that has been used to examine these relationships is the Biological Sensitivity to Context (BSC) theory, which is a developmental model that proposes that individuals differ in their level of susceptibility to their environments, both positive and negative, due to neurobiological differences that alter receptivity to environmental influence (Boyce & Ellis, 2005). From this perspective, individuals who are highly physiologically reactive to stress experience greater malleability in reaction to their environment as a result of their neurobiology (Boyce & Ellis, 2005; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011). Thus, this framework suggests that high reactivity would bring about beneficial outcomes in supportive environments and

maladaptive outcomes (e.g., internalizing symptoms, health problems) in negative, high-stress environments (Boyce & Ellis, 2005; Rudolph et al., 2011).

The BSC stress response is thought to develop as a result of both genetic and environmental factors throughout early development (Boyce & Ellis, 2005). In an evolutionary context, both high and low physiological reactivity may be considered adaptive, such that they allow for higher odds for survival through the fit between the profile and the environmental context (i.e., hypervigilance in highly reactive youth promotes a quicker response to danger while lower stress reactivity confers more implicit health advantages throughout life; Del Giudice et al., 2011; Obradović, 2012). According to the BSC theory, children with lower reactivity levels are expected to be less affected by environmental conditions (Boyce & Ellis, 2005; Ellis & Boyce, 2008; Obradović, 2012). Thus, low reactivity may offer protection from the negative effects of stress, while high reactivity confers the advantage of plasticity and adaptability (Boyce & Ellis, 2005; Ellis & Boyce, 2008; Obradović, 2012).

To date, BSC research has focused mainly on child and adolescent samples; this work has provided support for the BSC framework's predictions regarding stress reactivity and environmental contexts. In general, children who display high reactivity have shown greater vulnerability to adversity than their peers who are less reactive (Obradović & Boyce, 2009). For instance, one study (Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010) examined the effects of physiological stress reactivity on child outcomes (e.g. externalizing problems, school engagement, prosocial behavior) in the context of family adversity (e.g., marital discord, anger expression, financial stress).

Results indicated that, in comparison to their peers, children who demonstrated greater RSA withdrawal fared the worst in conditions of high family adversity and the best in conditions of low family adversity. Another study demonstrated that SCL-R moderated the relationship between marital conflict and internalizing problems for girls, such that, when compared to girls with low reactivity, girls with higher SCL-R showed the largest increase in internalizing problems when marital conflict was high, but the smallest increase when marital conflict was low (El-Sheikh, Keller, & Erath, 2007).

The evidence from these studies of the moderating effects of SCL-R and RSA-R is consistent with expectations based on the BSC theory. However, it is important to note that this evidence does not necessarily favor the BSC framework over the diathesis-stress model, in which it is proposed that some individuals, as a result of a vulnerability factor (e.g., genetic, physiological, behavioral), have a highly increased risk of experiencing adverse effects from an environmental stressor (Belsky & Pluess, 2009). In a diathesis-stress model, moderation would still occur but only at high levels of adversity, while at low levels of adversity, differences in adjustment between individuals with high or low reactivity would not be expected. Some of this ambiguity arises from the statistical methods chosen to analyze the data from these studies (for a review, see Roisman et al., 2012). Thus, it is imperative that researchers attempt to differentiate between evidence in support of BSC versus diathesis-stress models of individual risk. Therefore, if evidence emerged in the current study that was consistent with the predictions of BSC, I planned to employ follow-up analytic methods suggested by Roisman and colleagues (2012) in order to more clearly compare these two models in the proposed associations. The first of these

methods is the Regions of Significance (RoS) on X test, which tests whether the outcome and moderator are correlated at both ends of the distribution of the predictor; if so, findings support BSC over a diathesis-stress model. The second method that I would employ is the use of the proportion of interaction (PoI), which is the proportion of the total area in the interaction plots that is uniquely attributable a BSC response. PoI values close to 0.00 provide support for a diathesis-stress model, while values close to 0.50 support a BSC response.

Currently, research is in the nascent stages of demonstrating the applicability of the BSC theory to the context of peer victimization. One recent study in this area focused on reactivity in the HPA axis rather than ANS reactivity. However, as the research is limited, it is important to examine the findings across these various methodologies in order to assess whether BSC theory can help explain individual differences in the outcomes associated with peer victimization. In this study, only children with high anticipatory cortisol levels demonstrated higher levels of ruminative responses to a social stressor in the presence of victimization, posing a health risk; in contrast, children with high anticipatory cortisol levels demonstrated fewer ruminative responses in the absence of victimization, representing a health benefit (Rudolph et al., 2011). These findings provide initial support for assessing BSC in the context of peer victimization.

Preliminary evidence also indicates that ANS reactivity in response to stress may be indicative of BSC in studies of peer victimization. For instance, in one of the first studies of BSC to examine peer victimization as the adverse environmental event, Rudolph and colleagues (2010) reported that salivary alpha amylase (sAA), which is an

index in the saliva of SNS activity, moderated the relation between peer victimization and aggression; these findings support the idea that biological sensitivity to context, as assessed with ANS measures of stress reactivity, contributes to individual differences in adjustment outcomes (Obradović, 2012).

Research suggests that different patterns of interaction of the SNS and the PNS provide important information in the prediction of adjustment outcomes (El-Sheikh et al., 2009), and both systems must be examined in order to more fully understand the relationship between physiological reactivity and adjustment outcomes (Beauchaine, 2001). As the SNS and PNS can function together in a variety of ways, four profiles of stress system activity have been identified. *Reciprocal activation* occurs when one branch is activated while the other withdraws (Berntson et al., 1991; Del Giudice et al., 2011). Specifically, *reciprocal SNS activation* occurs when the SNS is activated and the PNS withdraws, and conversely, *reciprocal PNS activation* occurs when the PNS is activated and the SNS withdraws (Berntson et al., 1991). A small percentage of people demonstrate either *coactivation* patterns, where both systems are activated at the same time, or *coinhibition* patterns, where neither system is activated in the face of threat (Del Giudice et al., 2011; El-Sheikh et al., 2009). However, little research has examined how the two systems work together in moderating associations between adversity and developmental outcomes. Considering they operate in a parallel and frequently opposing fashion, it is important to examine them in concert in an effort to more fully understand how the SNS and PNS systems impact functioning (El-Sheikh et al., 2009).

Some preliminary evidence regarding the interactions between the SNS and PNS systems suggests that patterns of coinhibition and coactivation exacerbate risk for the development of externalizing behaviors in the context of marital conflict, while both forms of reciprocal activation serve protective functions (El-Sheikh et al., 2009). Alternatively, from a BSC perspective, researchers have hypothesized and subsequently provided evidence that suggests that a high SNS response (Quas, Bauer, & Boyce, 2004) and greater PNS withdrawal (Obradović et al., 2010) in the face of a stressor are both indicative of a BSC response (Belsky & Pluess, 2009; Ellis, Essex, & Boyce, 2005). Other evidence from outside the BSC framework also suggests that a high SNS response (Cummings, El-Sheikh, Kouros, & Keller, 2007; El-Sheikh et al., 2007; El-Sheikh, 2005) and greater PNS withdrawal (McLaughlin, Rith-Najarian, Dirks, & Sheridan, 2015; Obradović et al., 2010) in the face of a stressor exacerbate risk for negative outcomes. Moreover, one study has found preliminary evidence to support reciprocal sympathetic activation as indicative of a BSC response, suggesting that this is a feasible way to assess this theory (Lafko, Murray-Close, & Shoulberg, 2015). Thus, in order to further explore the interaction of the SNS and PNS systems in the context of peer victimization, the fourth goal of the current study was to examine whether reciprocal sympathetic activation (i.e., higher SCL-R and greater RSA withdrawal) would be the stress response profile reflecting BSC.

Thus far, minimal research has examined how a BSC response may be applied to the relationship between peer victimization and symptoms of anxiety and depression (see Rudolph et al., 2011 for an example). Therefore, the current study extends existing

research by using a BSC framework to examine variation in the adjustment outcomes (i.e., anxiety and depressive symptoms) associated with peer victimization. More specifically, this study seeks to examine whether adults who exhibit reciprocal SNS activation to stress fare best in the context of little peer victimization and worst in the context of high levels of peer victimization, as compared to less reactive adults. While limited previous work has examined these relationships in emerging adults, in one study highly stress-reactive college students demonstrated more psychosomatic symptoms and depressive symptoms in a high-stress condition, but lower than average symptoms in a low-stress condition (Gannon, Banks, Shelton, & Luchetta, 1989). These results, provide initial support for applying a BSC model within this age group.

Although sparse, some research indicates there may be potential gender differences in the human stress response. For instance, there have been differences found in HPA functioning for males and females, such that males show a higher HPA stress response (e.g., cortisol) to achievement stressors than females, while females have demonstrated a higher HPA stress response when facing social rejection (Stroud, Salovey, & Epel, 2002). Additionally, research suggests that, under stress, the male response is typically “fight-or-flight,” while the female response resembles “tend-and-befriend,” which is marked by protecting children and seeking affiliation and support with a group, likely due in part to the function of oxytocin (Taylor, 2006; Taylor et al., 2000). Taken together, this research suggests that there is the potential for gender differences to exist in the BSC response. For instance, in accordance with the research presented regarding the potential moderating roles of gender and physiological reactivity,

it is possible that females who display a high BSC response may be most reactive to relational victimization due to their relational social goals. Further, males who display a high BSC response may be most reactive to physical victimization as a result of their instrumental social goals. Thus, gender may moderate the BSC response to these two distinct forms of peer victimization. Therefore, the fifth and final goal of this study was to examine whether gender further moderated BSC effects on the association between forms of victimization and symptoms of depression and anxiety, in an exploratory fashion.

As research by Obradović, Bush, and Boyce (2011) demonstrated that physiological stress reactivity and its role in prediction of maladaptive outcomes can vary based on the nature of the stressor, it is important for the stressor task to be carefully selected by researchers. A study by Rudolph and colleagues (2011) found individual differences in physiological responses to a relational stress task moderated the relationship between peer victimization and depressive symptoms in children. In addition, the associations examined in the current study (i.e., peer victimization) were relational in nature. Thus, a relational stressor task was used to induce a stress response.

The Current Study

Aims & Hypotheses

The first goal of this study was to examine whether there was an association between physical and relational victimization and anxiety and depressive symptoms. I expected both physical and relational victimization to be positively associated with anxiety and depressive symptoms, and that relational victimization would be more

strongly associated with these outcomes (Hypothesis 1). The second goal of this study was to examine whether there were gender differences in the frequency of experiences of physical and relational victimization. I expected that females would demonstrate higher levels of relational victimization while males would demonstrate higher levels of physical victimization (Hypothesis 2). The third goal of this study was to examine whether gender moderated the proposed associations between victimization and anxiety and depressive symptoms. Given that males and females have been shown to react differently to relational and physical victimization (Crick, Grotpeter, & Bigbee, 2002; Crick & Nelson, 2002; Dempsey & Storch, 2008; Gabriel & Gardner, 1999; Leadbeater et al., 1999; Maccoby, 1990; Paquette & Underwood, 1999; Rudolph & Hammen, 1999; Rudolph, 2002; Rudolph et al., 2000; Shih et al., 2006), I expected the positive association between relational victimization and symptoms of anxiety and depression would be stronger for women than men (Hypothesis 3a), whereas the positive association between physical victimization and symptoms of anxiety and depression would be stronger for men than women (Hypothesis 3b).

To date, almost no BSC research has focused on the interaction between the SNS and the PNS. Thus, the fourth goal of the current study was to extend previous BSC research that investigated each system in isolation to examine whether reciprocal sympathetic activation (i.e., high SCL-R and RSA withdrawal) was indicative of a BSC response. I expected that victimization would be most strongly associated with anxiety and depressive symptoms among participants exhibiting reciprocal SNS activation, such that these individuals would exhibit the highest levels of anxiety/depression at high levels

of victimization and the lowest levels of anxiety/depression at low levels of victimization (Hypothesis 4). Finally, the fifth goal of this study was to examine whether gender moderated the BSC response to peer victimization. I expected that there may be potential gender differences in the role of the interaction between the SNS and PNS in moderating the associations between victimization and anxiety and depressive symptoms (Hypothesis 5). However, given the dearth of research on this association, the analyses related to this hypothesis were exploratory.

Method

Participants

Participants were drawn from a larger study examining the association between ANS activity and psychological functioning. The final sample for the present study included 246 emerging adults recruited from introduction to psychology courses at a northeastern public university. Participants were aged 18 to 23 years ($M_{age} = 18.77$, $SD = .97$) and were predominantly female (74%) and Caucasian (84%). Participants received course credit for their participation.

Procedure

Participants provided verbal consent and were familiarized with the laboratory and physiological equipment prior to completing a two-and-a-half-hour interview. Participants were attached to the physiological equipment in order to assess autonomic arousal and were asked to complete various stressor tasks (including the stress interview included in the present study), counterbalanced in order, as well as baseline measures of arousal. Participants were then asked to complete self-report measures of their

experiences of peer relational and physical victimization, their current anxiety and depressive symptoms, and additional questionnaires not included in the present study.

Assessment of Physiological Reactivity

Participants' physiological reactivity was assessed during a semi-structured interview during which they relived a recent stressful experience of relational victimization. This interview was an adapted version of the Social Competence Interview (SCI; Ewart & Kolodner, 1991) that was changed to focus specifically on stressors related to relational victimization. During the current procedure, participants were provided with 8 cards describing a particular type of relational stress (e.g., getting left out; someone gossiping about you; receiving the silent treatment), and were asked to choose the type of situation that caused them the most stress during the last few months. They were then asked to recreate the situation, through the description of the events, their thoughts, and their feelings. The SCI interview was preceded by a recovery period from the previous task lasting 4 minutes (if the SCI was not the first stressor, based on counterbalancing) and a resting baseline (e.g., sitting quietly) lasting 4 minutes. The interview itself lasted approximately 12 minutes. Prior to the administration of any stress tasks, there was a 5-minute accommodation period to the physiological equipment during which the equipment was placed on the participant and tested, allowing a consistent period of adjustment to the equipment prior to the first baseline period. There were two additional stressor tasks used in the protocol that were not included in the present study. Participants were asked play a round of Cyberball, an adapted version of an online ball-tossing game lasting approximately 4 minutes (Williams, Cheung, & Choi, 2000), which

is designed to mimic social exclusion experiences. Additionally, participants were asked to engage in a mental arithmetic task (e.g., subtracting the number 7 from a 3-digit number) while experiencing minor verbal harassment from the experimenter telling them to work harder and faster. In order to address the possibility of spillover of physiological arousal between the tasks, the order of tasks was counterbalanced, and there were 4-minute recovery periods after each task was completed, followed by a 4-minute resting baseline period before the next task began.

Autonomic arousal was assessed using a physiological measurement system developed by the James Long Company (Caroga Lake, NY). This system, including a specialized computer, Snapmaster software, and a custom-made bioamplifier, was used to collect physiological data continuously throughout the stressor tasks and baseline periods. In the current study, SCL was assessed with two Ag/AgCl electrodes attached to the intermediate phalanges of the index and ring fingers of the participant's non-dominant hand with double-sided adhesive collars with a 1-centimeter-diameter circle to contain the conductance gel. Participants washed and dried their hands prior to attaching the electrodes.

RSA was measured using an electrocardiogram (ECG). Participants placed electrodes on opposite sides of their torso, near the base of the ribcage, as well as a ground lead placed on the sternum. The ECG channel high-pass filter was set to 0.1 Hz and the low-pass filter was set to 1000 Hz. Cardiac inter-beat intervals (IBI) were measured as the time in milliseconds between the R-waves. Pneumatic bellows were placed around the participant's chest in order to assess respiration. The James Long

Company IBI Analysis System Program algorithm was used to identify R-waves, and RSA was calculated using the ‘peak-to-valley’ method (Grossman & Svebak, 1987). This calculation is based on the minimum IBI during inspiration and the maximum IBI during expiration. Additionally, the RSA calculation included both ECG and respiration measurements in order to control for the effects of respiration (Grossman, Karemaker, & Wieling, 1991).

In order to calculate SCL-R and RSA-R scores, mean levels of SCL and RSA were first calculated separately at baseline and during the stressor task. Baseline means were then subtracted from stressor task means to calculate both SCL-R and RSA-R. For SCL-R, positive values indicated increased reactivity levels, while negative values indicated decreased reactivity levels. For RSA-R, negative values indicated RSA withdrawal while positive values indicated RSA augmentation.

Measures

Peer Relational and Physical Victimization. Participants were administered the Self-Report of Aggression & Social Behavior Measure (SRASBM; Morales & Crick, 1998, unpublished manuscript) in order to assess self-reported physical and relational victimization. The two peer victimization subscales from this 59-item measure were used in the current study. The physical victimization subscale included three items that describe experiences of physical victimization (e.g., “I have been pushed or shoved by people when they are mad at me,” “I have a friend who has threatened to physically harm me in order to get his/her own way”). The second subscale assessed participants’ experiences of relational victimization. This subscale included four items describing

situations of relational victimization (e.g., “A friend of mine has gone ‘behind my back’ and shared private information about me with other people,” “I have a friend who excludes me from doing things with her/him and her/his other friends when s/he is mad at me”). For both subscales, participants rated how true each statement was for them currently and during the last year, using a 7-point Likert scale (1 = “not at all true” to 7 = “very true”; Cronbach’s α for physical victimization = .56; for relational victimization = .79). Scores for each subscale were then calculated by averaging the ratings across the three and four items, respectively.

Anxiety symptoms. Participants reported on their anxiety symptoms using the Beck Anxiety Inventory - Trait (BAIT; Beck & Steer, 1990), which is a 21-item measure assessing dispositional levels of anxiety. Participants were asked to rate how much each cognitive or somatic symptom (e.g., “unable to relax”; “hands trembling”) bothered them on a day-to-day basis using a 4-point Likert scale (0 = “not at all” to 3 = “severely”; Cronbach’s α = .90). Scores were calculated by taking the mean of the ratings of all the items. The BAIT has been shown to have favorable psychometric properties when used with college students (Kohn, Kantor, DeCicco, & Beck, 2008).

Depressive symptoms. Participants reported on their depressive symptoms using the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977), which is a 20-item measure assessing the frequency of symptoms of depression. Participants were asked to rate how frequently they experienced each symptom (e.g., “you thought your life had been a failure”; “you could not get ‘going’”) during the past week using a 4-point Likert scale (1 = “rarely or none of the time” to 4 = “most or all of the time”; Cronbach’s

$\alpha = .90$). Scores were calculated by taking the mean of the ratings of all the items. This measure has been shown to have favorable psychometric properties when used with college students (e.g., Radloff, 1991).

Results

Preliminary Analyses

Descriptive Analyses. Descriptive statistics for study variables are presented in Table 1. Based upon the recommendations outlined by Tabachnick and Fidell (2007) to evaluate violations of normality in the data (i.e., skewness or kurtosis), the variables in this study were assessed and violations were present for all variables. Thus, assumptions for normality were not met and therefore, as recommended by Russell and Dean (2000), bootstrapping techniques were used to address these violations in all correlation and regression analyses.

Missing data. Percentages of missing data for all study variables are presented in Table 2. Missing data levels of 5% or less are unlikely to result in biased estimates (Graham, 2009). Most of the variables in the current study had less than 5% missing data. The only variable missing more than 5% of data was RSA-R, with 9% of RSA-R scores missing. In order to examine whether missing data were missing completely at random, Little's Missing Completely at Random (MCAR) test was used. This test was not significant ($\chi^2(13) = 5.85, p > .05$), suggesting that these data were MCAR. Taking these results together, it appears that the missing data for the current study are largely ignorable.

Bootstrapping. Even when missing data are unlikely to bias findings, it is generally advised to address missing data using techniques such as multiple imputation (Graham, 2009). However, bootstrapping techniques and multiple imputation are not compatible when using SPSS 22 (IBM SPSS, Inc., 2013), and in the current study, it appeared that violations of normality were more likely to bias estimates than missing data. Therefore, bootstrapping techniques to accommodate violations of normality and listwise deletion to accommodate missing data were used for all correlations and hierarchical multiple regression analyses.

Bivariate Correlations. Intercorrelations between study variables are presented in Table 3. For each correlation, one thousand bootstrap samples were drawn with replacement, and the bias-corrected and accelerated (BCa) 95% confidence intervals were reported for all analyses. BCa confidence intervals are used to account for bias and skewness in the distribution of the standard errors in the bootstrap samples. Anxiety symptoms were significantly and positively associated with depressive symptoms ($r = .55$, BCa 95% CI: [.45 - .65]). Anxiety symptoms were also significantly and positively related to both relational victimization ($r = .23$, BCa 95% CI: [.11 - .35]) and physical victimization ($r = .18$, BCa 95% CI: [.04 - .33]). Depressive symptoms were also significantly and positively related to both relational victimization ($r = .30$, BCa 95% CI: [.17 - .43]) and physical victimization ($r = .23$, BCa 95% CI: [.08 - .37]). Relational victimization and physical victimization were significantly and positively associated with each other ($r = .37$, BCa 95% CI: [.24 - .49]). Relational victimization and gender were positively associated ($r = .16$, BCa 95% CI: [.05 - .28]), such that females experienced

more relational victimization than males, and physical victimization and gender were negatively associated ($r = -.16$, BCa 95% CI: $[-.32 - -.03]$), such that males experienced more physical victimization than females. Finally, physical victimization and RSA-R were positively associated ($r = .14$, BCa 95% CI: $[.02 - .31]$).

Primary Analyses

Bivariate Correlations. As expected (Hypothesis 1), correlational analyses indicated that both relational and physical victimization were positively associated with both anxiety symptoms and depressive symptoms. Contrary to Hypothesis 1, however, no significant differences were found in the strength of the relationships between the two forms of victimization and anxiety symptoms ($z = -.43$, $p > .05$) and depressive symptoms ($z = -1.03$, $p > .05$), respectively.

Repeated-Measures ANOVA. A repeated-measures ANOVA with form of aggression serving as the within-subjects factor and gender serving as the between-subjects factor indicated that there was a significant within-subjects effect of form of victimization ($F(1, 244) = 138.34$, $p < .001$), such that mean levels of relational victimization were greater than mean levels of physical victimization ($M_{rvic} = 2.88$, $M_{pvic} = 1.53$). There was not a significant between-subjects effect of gender ($F(1, 244) = .70$, $p > .05$). However, there was a significant interaction between type of victimization and gender ($F(1, 244) = 19.50$, $p < .001$). Follow-up repeated-measures ANOVAs investigating the frequency of relational and physical victimization within gender indicated that females demonstrated higher mean levels of relational victimization than physical victimization ($M_{rvic} = 3.01$, $M_{pvic} = 1.45$; $F(1, 183) = 236.23$, $p < .01$). Similarly,

males also demonstrated higher mean levels of relational victimization than physical victimization ($M_{rvic} = 2.47$, $M_{pvic} = 1.76$; $F(1, 61) = 25.65$, $p < .01$; see Figure 1).

However, as evidenced by the significant interaction between gender and form of victimization, the tendency to experience relational victimization at higher rates than physical victimization was more pronounced for women than for men.

Hierarchical Multiple Regression Analyses. For each regression, one thousand bootstrap samples were drawn with replacement. For findings that approached conventional levels of statistical significance in the first analysis, analyses were re-run using either two thousand or five thousand bootstrap samples and a random number seed. This was to minimize sampling error and to provide estimates that replicate across runs, given the variability in estimates that emerge based on the random resampling process when using ~~bootstrapping~~bootstrapping techniques (Hayes, 2013). The unstandardized regression coefficients, bias (i.e., the difference between the sample unstandardized regression coefficient and the values of the average unstandardized regression coefficient across the bootstrap samples), the bootstrap standard error (i.e., the standard error averaged across the bootstrap samples), and the bias-accelerated and corrected (BCa) 95% confidence intervals are reported for all analyses. According to the procedures outlined by Aiken and West (1991), all continuous predictors were mean-centered.

Results for the models examining the associations between victimization (i.e., relational and physical victimization) and anxiety symptoms and depressive symptoms, moderated by gender, are presented in Tables 4, 5, 6, and 7. In these models, the non-focal form of victimization was entered in the first step. There is theoretical and empirical

overlap in the constructs of relational and physical victimization (Cole, Maxwell, Dukewich, & Yosick, 2010); thus, it is important in this analysis to separate out the variance associated with each form in order to investigate the unique predictive power of each. In the second step, the focal form of victimization was entered along with gender, which was coded using effect coding (-1 = males and 1 = females). Finally, the two-way interaction between the focal form of victimization and gender was entered. Contrary to Hypotheses 3a and 3b, neither the two-way interactions between relational victimization and gender in the prediction of anxiety symptoms ($b = -.025$, BCa 95% CI: [-.060 - .013]) or depressive symptoms ($b = -.051$, BCa 95% CI: [-.115 - .014]), nor the two-way interactions between physical victimization and gender in the prediction of anxiety symptoms ($b = .011$, BCa 95% CI: [-.069 - .088]) or depressive symptoms ($b = .027$, BCa 95% CI: [-.082 - .134]) were significant.

The second set of regression analyses investigated the moderating effects of SCL-R and RSA-R on the relationship between relational and physical victimization and anxiety and depressive symptoms, respectively (Tables 8, 9, 10, and 11). In these models, the non-focal form of victimization was entered in the first step, along with gender because it was shown to be correlated with both forms of victimization. In the second step, the focal form of victimization was entered along with SCL-R and RSA-R. Next, the two-way interactions involving the focal form of victimization, RSA-R and SCL-R were entered. In the fourth step, the three-way interaction between the focal form of victimization, SCL-R, and RSA-R was entered. Contrary to Hypothesis 4, the three-way interaction between relational victimization, SCL-R, and RSA-R in the prediction of

anxiety symptoms ($b = .357$, BCa 95% CI: [-.438 – 1.173]), along with the three-way interactions between physical victimization, SCL-R, and RSA-R in the prediction of anxiety symptoms ($b = .741$, BCa 95% CI: [-.945 - 3.875]) and depressive symptoms ($b = .757$, BCa 95% CI: [-.806 - 2.142]), were not significant. However, the three-way interaction between relational victimization, SCL-R, and RSA-R in the prediction of depressive symptoms was marginally significant ($b = .780$, BCa 95% CI: [-.005 – 1.330]). Follow-up analyses, however, were not consistent with the hypothesis that participants exhibiting reciprocal SNS activation would exhibit the highest levels of anxiety/depressive symptoms at high victimization and the lowest levels of these symptoms at low victimization (Hypothesis 4). Instead, as depicted in Figure 2, simple slope analyses indicated that relational victimization was significantly and positively associated with depressive symptoms among individuals demonstrating physiological coactivation ($t = 3.02$, BCa 95% CI: [.045 – .215]) and coinhibition ($t = 3.03$, BCa 95% CI: [.056 – .263]). The simple slopes were not significant for individuals exhibiting reciprocal SNS activation ($t = .75$, BCa 95% CI: [-.070 – .156]) or reciprocal PNS activation ($t = -.04$, BCa 95% CI: [-.084 – .080]). Finally, the methods suggested by Roisman and colleagues to compare BSC and diathesis-stress models (2012; i.e., RoS, PoI) were not employed in this study, as the significant profiles of reactivity were not reflective of a BSC response.

The third set of analyses were exploratory and investigated the moderating effects of gender, SCL-R, and RSA-R on the relationship between relational and physical victimization and anxiety and depressive symptoms, respectively (Tables 12, 13, 14, and

15). In these models, the non-focal form of victimization was entered in the first step. In the second step, the focal form of victimization was entered along with SCL-R, RSA-R and gender. In the third step, the six two-way interactions involving the focal form of victimization, SCL-R, RSA-R, and gender were entered. In the fourth step, the four three-way interactions were entered. Finally, the four-way interaction was entered in the fifth step of the model. Neither the four-way interactions between relational victimization, SCL-R, RSA-R, and gender in the prediction of anxiety symptoms ($b = -.414$, BCa 95% CI: [-1.889 – 1.236]) or depressive symptoms ($b = -.338$, BCa 95% CI: [-2.310 – 2.640]), nor the four-way interactions between physical victimization, SCL-R, RSA-R, and gender in the prediction of anxiety symptoms ($b = -1.339$, BCa 95% CI: [-5.872 – 1.720]) or depressive symptoms ($b = .920$, BCa 95% CI: [-3.708 – 5.142]) were significant.

Follow-up Analyses. In the analyses examining the moderating effects of gender, SCL-R and RSA-R on the association between relational victimization and anxiety symptoms, a significant three-way interaction emerged in Step 4 that required further analysis (see Table 12). It is important to assess these interactions in isolation, in order to examine whether they only emerged as significant in models controlling for other interactions. This follow-up analysis examined the interaction between gender, SCL-R and RSA-R in the prediction of anxiety symptoms. A follow-up multiple regression revealed that this interaction was not significant when considered in isolation (Table 16).

Discussion

The overall goal of the current study was to investigate whether relational and physical victimization were related to anxiety and depressive symptoms and if gender and ANS reactivity to relational victimization stressors moderated these relationships in a population of emerging adults. Findings suggested that both physical and relational victimization were related to both anxiety and depressive symptoms in this population. Additionally, females were more likely to experience relational victimization than males, while males were more likely to experience physical victimization than females. Finally, relational victimization was significantly related to depressive symptoms in individuals demonstrating coactivation and coinhibition patterns of stress reactivity, although the interaction only approached conventional levels of statistical significance.

Type of victimization and adjustment in emerging adulthood

The first goal of the current study was to examine the unique associations between both physical and relational victimization and internalizing symptoms (i.e., symptoms of anxiety and depression) and potential differences in the strength of these associations in a sample of emerging adults. Consistent with Hypothesis 1, both physical and relational victimization were positively associated with symptoms of both anxiety and depression. These findings extend previous research regarding the negative correlates of peer victimization conducted in samples of children and adolescents (Cullerton-Sen & Crick, 2005; Gros et al., 2010; Rudolph et al., 2009; Troop-Gordon & Ladd, 2005). Despite the potential for victimization experiences to influence adjustment outcomes in emerging adults, research on these associations is lacking in this age group (Heilbron &

Prinstein, 2008). Primarily, the focus of prior research that has linked peer victimization experiences with anxiety and depressive symptoms has been on children ranging from preschool-age through adolescence (e.g., Boivin et al., 1995; Crick & Bigbee, 1998; Crick et al., 1999; Crick & Grotpeter, 1995; Crick & Nelson, 2002; Gros et al., 2010; Hodges & Perry, 1999; Klomek et al., 2008; Prinstein et al., 2001; Rudolph et al., 2009; Troop-Gordon & Ladd, 2005; Vuijk et al., 2007). The limited research exploring the correlates of peer victimization during emerging adulthood is notable; social relationships are granted greater importance as children enter adolescence (Parker et al., 2006), thus, victimization experiences may become a critical marker of adjustment during this time and throughout the transition to adulthood. Research that has been conducted on emerging adults suggests that the negative effects of victimization can last through adolescence and early adulthood (Gros et al., 2010; Kumpulainen et al., 1999; Roth et al., 2002). However, most of what is known about these processes is based on research in younger samples. Thus, the current study extended this previous work, suggesting that the positive associations between physical and relational victimization and internalizing outcomes (e.g., anxiety and depressive symptoms) are evident in the later developmental period of emerging adulthood. Therefore, these social processes appear relevant and potentially damaging during early adulthood.

I also hypothesized that relational victimization would be more strongly associated with symptoms of anxiety and depression than physical victimization (Hypothesis 1). Contrary to expectations, the strength of the associations between victimization and anxiety and depressive symptoms, respectively, did not differ by form

of victimization. It is interesting to note, however, that in the hierarchical multiple regression analyses, relational victimization was uniquely associated with the outcomes, when controlling for physical victimization. In contrast, physical victimization was not uniquely associated with the outcomes, above and beyond relational victimization. Therefore, although relational victimization was not statistically more strongly related to the outcomes than physical victimization, relational victimization appears to provide unique information about who is at risk, above and beyond what can be learned from evaluating physical victimization. In contrast, physical victimization did not provide additional information regarding risk for internalizing pathology, once relational victimization was controlled. These findings highlight the importance of assessing relational victimization when investigating risk factors for the development of anxiety and depressive symptoms.

Gender

The second goal of this study was to examine whether there were gender differences in the frequency of experiences of physical and relational victimization. Consistent with Hypothesis 2, females experienced higher levels of relational victimization than males did. Similarly, males experienced higher levels of physical victimization than females did. Research has suggested that females may face more relational victimization based on their social goals of building and maintaining relationships (Dempsey & Storch, 2008; Paquette & Underwood, 1999; Rose & Rudolph, 2006; Rudolph et al., 2009), which leave them vulnerable to relational aggression from peers. On the other hand, distressing social situations for males tend to involve physical

dominance, peer status, competition, and instrumental concerns (Crick, Grotpeter, & Bigbee, 2002; Gabriel & Gardner, 1999; Maccoby, 1990; Rudolph, 2002; Shih et al., 2006). Therefore, males may be more likely to face behaviors from peers that target these goals, like physical victimization. However, it is also important to note that both males and females experienced higher levels of relational victimization than physical victimization (though this difference was more pronounced for females than for males). Thus, while gender differences did emerge in the current study in the frequency of experiences of physical and relational victimization, the differences were tempered by both men and women experiencing higher levels of relational, as compared to physical, victimization.

The third goal of this study was to examine whether gender moderated the proposed associations between victimization and anxiety and depressive symptoms. Contrary to expectations (i.e., Hypothesis 3a and 3b), there were no gender differences in the associations between relational victimization and physical victimization and anxiety and depressive symptoms, respectively. These findings contrast with some previous research and theory developed primarily with children and adolescents suggesting that males and females may respond differently to experiences of victimization (Crick & Nelson, 2002; Dempsey & Storch, 2008; Paquette & Underwood, 1999; Prinstein et al., 2001; Rudolph, 2002). This previous work has suggested that relational victimization may be more likely to lead to negative outcomes for females because this form of victimization effectively harms females' relationship-oriented social goals (Crick & Bigbee, 1998). Similarly, the status- and dominance-oriented nature of males' social

goals may put them at greater risk for negative outcomes when they face experiences such as physical victimization that directly target their highly valued social goals.

As the theoretical and empirical work in this area has focused on younger samples, the failure to find gender moderation in the current study may reflect developmental differences in social goals. Specifically, gender differences that are evident in earlier age groups may not carry forward into emerging adulthood. For instance, social changes that occur during the transition to college (e.g. living in dorms) may lead to relatively similar social goals in males and females, as compared to earlier developmental periods. In fact, some research has documented smaller gender differences in social goals in emerging adulthood than in adolescence; specifically, LaFontana and Cillessen (2010) reported that the gender difference in the priority of status over friendship was greatest during high school and declined into emerging adulthood. Emerging adulthood may be a time when the gender differences in social goals are fading. With more similar social goals, males and females may be more likely to react similarly to experiences of physical and relational victimization during this developmental period. Future research should focus on longitudinal studies that can explore developmental differences in social goals, and whether these changes have implications for the correlates of physical and relational victimization in males and females.

Alternatively, the failure to find gender moderation may reflect low statistical power in moderation analyses. In the present study, 75% of the sample was female, limiting the power to detect differences between genders. In addition, few individuals

highly endorsed experiences of relational victimization (0% of males and 5% of females reported a 6 or above on a 7-point scale) or physical victimization (0% of males and 0% of females). This lack of variability may limit the power to detect gender differences in the strength of the associations between peer victimization and adjustment outcomes (e.g., anxiety or depressive symptoms). It is imperative that future research include a larger sample size overall, with a higher proportion of male participants. Additionally, investigating these processes in a higher risk sample (e.g., by recruiting groups of highly victimized individuals) will be important for clarifying the relationships presented in the current study.

Autonomic Reactivity

The fourth goal of the current study was to extend previous BSC research that investigates the SNS and PNS in isolation to examine whether reciprocal sympathetic activation (i.e., high SCL-R and greater RSA withdrawal) was indicative of a BSC response. Contrary to Hypothesis 4, those experiencing reciprocal sympathetic activation in the face of the stressor task did not demonstrate the strongest relationships between victimization (physical or relational) and anxiety or depressive symptoms. For physical victimization, the moderating role of the interaction between SCL-R and RSA-R in the association between victimization and both anxiety and depressive symptoms was not significant. For relational victimization, the moderating role of the interaction between SCL-R and RSA-R in the association between victimization and anxiety was also not significant; however, the 3-way interaction between relational victimization, SCL-R, and RSA-R was a marginally significant predictor when the outcome was depressive

symptoms. Unexpectedly, follow-up analyses indicated that relational victimization was positively associated with depressive symptoms among individuals who exhibited *coactivation* and *coinhibition*, rather than those who exhibited a *reciprocal SNS response*. These findings were unexpected under the framework of the BSC theory, which suggests that a high SNS response (Quas et al., 2004) and greater PNS withdrawal (Obradović et al., 2010) in the face of a stressor would both be indicative of a BSC response (Belsky & Pluess, 2009; Ellis et al., 2005).

However, BSC theory is just one of several existing perspectives regarding how physiological reactivity may moderate the associations between adversity and adjustment outcomes. El-Sheikh and colleagues (2009) have offered an alternative perspective, suggesting that patterns of coinhibition and coactivation may both exacerbate risk for negative outcomes in the context of adversity. These authors argue that coinhibition and coactivation may denote that the body is experiencing a breakdown in regulation, with either the SNS or the PNS failing to perform adequately in response to stress (El-Sheikh & Erath, 2011; El-Sheikh et al., 2009). For instance, during a coactivation response, the SNS is activated. This SNS activation represents the initiation of physiological arousal and the mobilization of metabolic resources to engage with the stressor (Obradović, 2012; Sijtsma et al., 2011). However, the implications of this resource mobilization may depend on whether the PNS response is adaptive. Greater RSA withdrawal in response to a stressor is hypothesized to reflect strong emotion regulation capabilities and has been linked with lower levels of internalizing symptoms (Graziano & Derefinko, 2013). Blunted RSA withdrawal (or RSA augmentation), in contrast, may lead to impaired

emotion regulation capabilities and coping difficulties (Graziano & Derefinko, 2013; Hinnant & El-Sheikh, 2009). Therefore, when individuals experience coactivation, the lack of emotion regulation from the PNS combined with the physiological arousal from the SNS may lead to dysregulated emotional reactivity, potentially resulting in negative outcomes and a failure to cope effectively with the stressor (e.g., victimization; El-Sheikh et al., 2009; Wagner & Abaied, 2015). As such, responses to peer victimization may be maladaptive in nature and ultimately increase risk for internalizing problems.

During a coinhibition response, the individual experiences PNS withdrawal, which increases attention (Porges, 2007; Wagner & Abaied, 2015). This is coupled with a failure of the SNS to activate, which may prevent sufficient metabolic input to allow for effective behavioral self-regulation (El-Sheikh et al., 2009; Wagner & Abaied, 2015). This pattern may result in increased attention to the threat, but without input from the SNS, effective coping responses may be impaired (Wagner & Abaied, 2015). For instance, this pattern may represent a passive response to stress and lead to a failure to use active coping techniques (El-Sheikh, Keiley, Erath, & Dyer, 2013). This failure to sufficiently cope with stressful situations may exacerbate risk for internalizing symptoms in the context of high adversity.

There is some support for this theory in the literature to date. For instance, in one series of studies conducted with school-age children, patterns of coinhibition and coactivation exacerbated risk for the development of externalizing behaviors in an environment of marital conflict, while both forms of reciprocal activation served protective functions (El-Sheikh et al., 2009). Additional evidence from a study on

emerging adults suggests that relational victimization predicted reactive relational aggression in individuals demonstrating coinhibition and coactivation responses (Wagner & Abaied, 2015). This provides support for the moderating role of coactivation and coinhibition profiles in the relationship between risk and negative adjustment outcomes such as externalizing pathology. However, findings have been mixed in studies of internalizing outcomes, for which reciprocal PNS activation may serve as a greater risk factor (El-Sheikh et al., 2013). In addition, research in this area is still in the nascent stages, highlighting the importance of continued research on the roles of the interaction of the SNS and PNS on the relationships between adversity, such as peer victimization, and outcomes such as anxiety and depressive symptoms.

It is important to note that the roles of coactivation and coinhibition in moderating associations between victimization and adjustment was found only for relational victimization as the predictor and depressive symptoms as the outcome, and that it was only marginally significant. In the other relationships examined in the current study, physiological reactivity did not serve as a moderator. There are several possible explanations for this specificity of effects. It may be that relational victimization, rather than physical victimization, was a marginally significant predictor due to the relational nature of stressor used to elicit physiological reactivity (i.e., the SCI). Future studies could use a similar version of the SCI that focuses on the experience of physical, rather than relational, forms of peer victimization, as physiological stress reactivity and its role in the prediction of maladaptive outcomes has been shown to vary based on the nature of the stressor (Obradović et al., 2011). Additionally, there may be something unique about

how physiological reactivity functions in moderating the relationship between relational victimization and depressive symptoms that is not found for physical victimization. Relational victimization has been linked repeatedly to internalizing symptoms in the literature (Gros et al., 2010; Troop-Gordon & Ladd, 2005), while the relationships involving physical victimization have been less clear (Cullerton-Sen & Crick, 2005). Finally, the low internal consistency of the physical victimization measure ($\alpha = .56$) may additionally explain the pattern of findings in this study.

Interestingly, ANS reactivity moderated the association between relational victimization and symptoms of depression but not anxiety. These results are surprising, as victims often report elevated symptoms of both anxiety and depression (Boivin et al., 1995; Craig, 1998; Grills & Ollendick, 2002; Nishina et al., 2005; Paul & Cillessen, 2003; Slee, 1994; Slee, 1995). Moreover, these associations have been hypothesized to result from negative self-evaluations (Crick & Bigbee, 1998; Vuijk et al., 2007) or social isolation (Hanish & Guerra, 2002; Roth et al., 2002) among victimized youth, suggesting similar processes for each outcome. However, the findings of the current study raise the possibility that the processes linking victimization to anxiety and depressive symptoms, respectively, are different, at least in the context of physiological coactivation and coinhibition. It is possible, for instance, that depressive symptoms are more likely than anxiety symptoms to develop as a result of dysregulated stress reactivity to victimization. Rudolph and colleagues (2000) suggest that depressive symptoms, specifically, are often associated with interpersonal stressors, which may help explain the pattern of findings in

the current study. However, future research is needed in order to further examine this possibility.

The final goal of the present study was to examine whether gender moderated the BSC response to peer victimization. Contrary to Hypothesis 5, gender was not found to interact with physiological stress responses in moderating the associations between victimization and anxiety/depressive symptoms. While previous research suggested that there may be gender differences in the physiological stress response (Stroud et al., 2002; Taylor, 2006; Taylor et al., 2000), it is possible that males and females with specific physiological profiles may respond to victimization in the same way. Again, however, statistical power to detect gender differences was limited in this study, as the majority of the sample was female.

Strengths, Limitations & Future Directions

There were several strengths to the current study that contribute to a greater understanding of how peer victimization is associated with anxiety and depressive symptoms in emerging adults. First, the study employed two different measures of physiological stress reactivity (SCL-R and RSA-R), allowing an examination of how these two systems function individually and together, which has been identified as an important goal in this research domain (Beauchaine, 2001; El-Sheikh et al., 2009). Second, the stressor task used to elicit physiological reactivity was highly relevant to the variables of interest in this study. Third, the current study employed multiple levels of analysis, including self-report measures and indices of physiological functioning. Fourth, and finally, the current study employed confidence intervals in reporting statistical

significance, an approach that has been shown to avoid biases inherent in null hypothesis testing (Cohen, 1994).

There were several limitations to the current study. First, the study utilized self-report measures of the experiences of peer victimization and anxiety and depressive symptoms. It is possible that participants may have under- or over-reported instances of victimization as well as their experiences of symptoms in order to appear in a more favorable light. The low internal consistency of the physical victimization subscale suggests that it may not have reliably measured this construct in this sample.

Additionally, shared method variance is a concern in this study, such that an association between victimization and anxiety or depressive symptoms may be a result of using the same reporter for both constructs. Nevertheless, self-reports do confer the advantage of capturing instances of victimization that are known only to the individual experiencing them (Crick & Bigbee, 1998). Self-reports of internalizing symptoms also provide a strong methodology for understanding internal experiences that may not be visible or obvious to others. A further methodological concern is that this study examined only two measures within the ANS, which may limit findings. It may be important to include other measures of the ANS (e.g., sAA, blood pressure), as well as measures across other stress systems such as the HPA axis (e.g., cortisol), in order to fully examine how physiological reactivity impacts the outcomes of interest.

Importantly, the design of the current study was cross-sectional in nature, which limited the ability to determine the causality and directionality of the findings. There is much evidence to support the direction of effects examined in this study. Research has

shown that victimized children are at risk for developing anxiety and depression symptoms (Craig, 1998; Grills & Ollendick, 2002; Reijntjes, et al., 2010; Slee, 1995), perhaps because difficult peer interactions result in negative self-evaluations and potential isolation (Crick & Bigbee, 1998; Hanish & Guerra, 2002; Vuijk et al., 2007). However, there is also evidence to suggest that the relationship between victimization and internalizing problems is reciprocal, such that symptoms of anxiety and depression can lead to increased peer victimization (Crick & Bigbee, 1998; Vuijk et al., 2007). For instance, behaviors related to internalizing symptoms may be seen by bullies as signals that children cannot defend themselves, making them more vulnerable to victimization by peers (Hodges & Perry, 1999). A meta-analysis of longitudinal studies found support for both victimization predicting internalizing problems as well as internalizing problems predicting future victimization (Reijntjes et al., 2010). Future studies employing longitudinal designs will help to clarify the direction of effects, as well as any moderating constructs that may impact these relationships.

There are additional limitations based on the variable-centered nature of the analyses. Some research has found differences in the associations between victimization and internalizing symptoms based on whether the analyses were person-centered or variable-centered. For example, while the longitudinal study by Hanish and Guerra (2002) found support for victimization predicting internalizing symptoms, evidence did not support this relationship when they employed person-centered analyses. More specifically, members of the internalizing group (defined by high levels on the Child Behavior Checklist [CBCL] on the anxious/depressed and withdrawn subscales) tended

to experience relatively low levels of victimization at both time points and victimization did not predict inclusion in the group. However, the bulk of the evidence in this area, including variable-centered analyses from this longitudinal study, does suggest a link between peer victimization and internalizing symptoms. Thus, the current study should be extended through future research employing person-centered analyses in order to more fully understand the examined relationships.

Finally, there are limitations based on the demographics of our sample. First, there were more females than males in the current sample, which likely limited the ability to meaningfully understand the relationships in the proposed analyses for males, as well as the power to test gender moderation. Second, the sample was almost entirely Caucasian, which limited the generalizability of any findings to other racial groups. While I would expect similar processes across racial groups, further research is needed in order to test this. Additionally, the sample was drawn from a university population. It is not clear whether there would be differences between our sample and adults in this age group that are not enrolled in a university; thus, further research is needed in order to test this and the generalizability of my findings may be limited.

There are several implications from the current study. First, although a number of studies have examined the relationship between peer victimization and internalizing symptoms, the literature is limited in a couple of key areas. Many studies have focused solely on physical forms of peer victimization, or have failed to distinguish between the types, instead utilizing an over-arching peer victimization measure (e.g., Boivin et al., 1995; Hodges & Perry, 1999; Troop-Gordon & Ladd, 2005). The current study strived to

address this limitation by including separate measures of physical and relational victimization. This is particularly important because, in the present study, relational, but not physical, victimization was uniquely associated with the outcome measures.

Second, this study is innovative in the application of the interaction of the SNS and PNS as a moderator of the relationship between victimization and the outcomes of anxiety and depressive symptoms. While the proposed application of the Biological Sensitivity to Context theory to these relationships was not supported, coactivation and coinhibition profiles of reactivity were found to be significant moderators of the association between relational victimization and depressive symptoms. This provides important information in the further development of theories pertaining to the moderating role of physiological reactivity in the relationships between adversity and negative outcomes.

Third, research on how the relationships between peer victimization and anxiety and depressive symptoms is moderated by physiological reactivity is lacking in the emerging adult population. Thus, the current study provided valuable insight into how these relationships function at a different point in the lifespan than what is most commonly investigated. This information may add in the development of future intervention and prevention efforts.

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Table 1.

Means, Standard Deviations, and Normality Statistics

Variable	Mean	SD	Range	Skewness (S.E.)	Kurtosis (S.E.)
1. Age	18.77	.97	18.00 – 23.00	1.52 (.16) ***	2.43 (.31) ***
2. Gender	.50	.87	-1.00 – 1.00	-1.15 (.16) ***	-.69 (.31) ***
3. AS	1.40	.37	1.00 – 3.30	1.56 (.16) ***	3.20 (.31) ***
4. DS	1.71	.48	1.00 – 3.30	.97 (.16) ***	.47 (.31) ***
5. RV	2.88	1.44	1.00 – 7.00	.71 (.16) ***	-.22 (.31) ***
6. PV	1.53	.80	1.00 – 5.67	2.00 (.16) ***	4.55 (.31) ***
7. SCL-R	1.72	1.51	-1.20 – 10.34	1.77 (.16) ***	5.33 (.31) ***
8. RSA-R	-.01	.05	-.43 - .30	-1.66 (.16) ***	26.58 (.32) ***

Notes: AS = Anxiety Symptoms. DS = Depressive Symptoms. RV = Relational Victimization. PV = Physical Victimization. SCL-R = Skin Conductance Level Reactivity. RSA-R = Respiratory Sinus Arrhythmia Reactivity. Gender coded: -1 = Male, 1 = Female.

*** $p \leq .01$

Table 2.

Overview of missing data for study variables

Variable	Cases missing data	Percentage of data missing
1. Age	3	1.0%
2. Gender	3	1.0%
3. AS	3	1.0%
4. DS	3	1.0%
5. RV	3	1.0%
6. PV	3	1.0%
7. SCL-R	6	2.0%
8. RSA-R	23	9.0%

Notes: AS = Anxiety Symptoms. DS = Depressive Symptoms. RV = Relational Victimization. PV = Physical Victimization. SCL-R = Skin Conductance Level Reactivity. RSA-R = Respiratory Sinus Arrhythmia Reactivity.

Table 3.
Intercorrelations among Study Variables

	1	2	3	4	5	6	7	8
1. Age	1							
2. Gender	-.09 (-.21 - .05)	1						
3. AS	-.02 (-.13 - .11)	.10 (-.03 - .23)	1					
4. DS	.09 (-.03 - .21)	.01 (-.14 - .16)	.55 (.45 - .65)	1				
5. RV	-.07 (-.18 - .06)	.16 (.05 - .28)	.23 (.11 - .35)	.30 (.17 - .43)	1			
6. PV	.04 (-.08 - .16)	-.16 (-.32 - -.03)	.18 (.04 - .33)	.23 (.08 - .37)	.37 (.24 - .49)	1		
7. RSA-R	-.01 (-.12 - .10)	.02 (-.09 - .14)	.07 (-.06 - .22)	.07 (-.09 - .30)	.08 (-.06 - .26)	.14 (.02 - .31)	1	
8. SCL-R	.02 (-.11 - .15)	-.02 (-.16 - .13)	-.10 (-.20 - .01)	-.08 (-.19 - .03)	.00 (-.11 - .11)	.11 (-.04 - .26)	.03 (-.12 - .14)	1

Notes: AS = Anxiety Symptoms. DS = Depressive Symptoms. RV = Relational Victimization. PV = Physical Victimization.

RSA-R = Respiratory Sinus Arrhythmia Reactivity. SCL-R = Skin Conductance Level Reactivity.

Gender coded: -1 = Male, 1 = Female.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

Parenthetical notation indicates BCa 95% confidence interval (bold font indicates statistical significance at $p \leq .05$)

Table 4.

Regression Models for Relational Victimization and Gender Predicting Anxiety Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% <i>CI</i>	ΔR^2
1	PV	.079 (.001, .035)	.011 - .150	.029**
2	Gender	.042 (.000, .028)	-.014 - .096	.030*
	RV	.034 (.001, .017)	.002 - .069	
3	RV x Gender	-.025 (.001, .018)	-.060 - .013	.005

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization.

Gender coded: -1 = Male, 1 = Female.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 5.
Regression Models for Relational Victimization and Gender Predicting Depressive Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% <i>CI</i>	ΔR^2
1	PV	.141 (.003, .053)	.045 - .263	.056***
2	Gender	.006 (.003, .038)	-.073 - .092	.054***
	RV	.082 (.001, .022)	.040 - .129	
3	RV x Gender	-.051 (.001, .030)	-.115 - .014	.014*

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization.

Gender coded: -1 = Male, 1 = Female.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 6.

Regression Models for Physical Victimization and Gender Predicting Anxiety Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% <i>CI</i>	ΔR^2
1	RV	.051 (.001, .015)	.022 - .081	.039**
2	Gender	.042 (.000, .028)	-.014 - .096	.019 ⁺
	PV	.063 (.001, .040)	-.011 - .144	
3	PV x Gender	.011 (.000, .041)	-.069 - .088	.000

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization.

Gender coded: -1 = Male, 1 = Female.

⁺ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 7.

Regression Models for Physical Victimization and Gender Predicting Depressive Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% <i>CI</i>	ΔR^2
1	RV	.101 (.001, .022)	.055 - .152	.093***
2	Gender	.006 (.003, .038)	-.073 - .092	.017
	PV	.086 (.003, .055)	-.015 - .202	
3	PV x Gender	.027 (.000, .057)	-.082 - .134	.001

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization.

Gender coded: -1 = Male, 1 = Female.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 8.

Regression Models for Relational Victimization, SCLR, and RSAR Predicting Anxiety Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% CI	ΔR^2
1	PV	.093 (.002, .040)	.016 - .178	.051***
	Gender	.058 (.001, .026)	.002 - .109	
2	RV	.039 (.000, .019)	.001 - .075	.034*
	SCL-R	-.027 (.000, .014)	-.055 - .002	
	RSA-R	.267 (.010, .441)	-.616 - 1.58	
3	RV x RSA-R	-.119 (-.061, .424)	-.919 - .525	.002
	RV x SCL-R	.004 (.000, .013)	-.020 - .032	
	SCL-R x RSA-R	-.201 (.026, .350)	-.885 - .656	
4	RV x SCL-R x RSA-R	.357 (.050, .313)	-.438 - 1.173	.008

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization, SCL-R = skin conductance reactivity, RSA-R = respiratory sinus arrhythmia reactivity.

Gender coded: -1 = Male, 1 = Female.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 9.

Regression Models for Relational Victimization, SCLR, and RSAR Predicting Depressive Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% CI	ΔR^2
1	PV	.135 (.005, .054)	.044 - .265	.054**
	Gender	.024 (.000, .041)	-.059 - .105	
2	RV	.079 (-.001, .023)	.038 - .120	.063**
	SCL-R	-.029 (.000, .017)	-.064 - .006	
	RSA-R	.312 (.181, .791)	-.683 - 2.761	
3	RV x RSA-R	-.340 (.033, .717)	-1.621 - 1.265	.006
	RV x SCL-R	.015 (-.001, .017)	-.015 - .043	
	SCL-R x RSA-R	-.051 (.080, .588)	-1.036 - 1.411	
4	RV x SCL-R x RSA-R	.780 (-.083, .425)	-.005 - 1.330	.025**

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization SCL-R = skin conductance reactivity, RSA-R = respiratory sinus arrhythmia reactivity.

Gender coded: -1 = Male, 1 = Female.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 10.

Regression Models for Physical Victimization, SCLR, and RSAR Predicting Anxiety Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% CI	ΔR^2
1	RV	.055 (.000, .015)	.024 - .086	.056**
	Gender	.029 (.000, .026)	-.025 - .087	
2	PV	.068 (.000, .042)	-.006 - .155	.029 ⁺
	SCL-R	-.027 (.001, .015)	-.060 - .006	
	RSA-R	.267 (.011, .448)	-.564 - 1.303	
3	PV x RSA-R	-1.308 (-.133, .965)	-2.881 - .110	.019
	PV x SCL-R	-.019 (.002, .019)	-.048 - .033	
	SCL-R x RSA-R	-.236 (.136, .385)	-.875 - 1.088	
4	PV x SCL-R x RSA-R	.741 (.284, .792)	-.945 - 3.875	.009

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization, SCL-R = skin conductance reactivity, RSA-R = respiratory sinus arrhythmia reactivity.

Gender coded: -1 = Male, 1 = Female.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

⁺ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 11.

Regression Models for Physical Victimization, SCLR, and RSAR Predicting Depressive Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% CI	ΔR^2
1	RV	.099 (.000, .024)	.024 - .086	.093***
	Gender	-.024 (.002, .039)	-.106 - .061	
2	PV	.080 (.005, .055)	-.028 - .216	.024
	SCL-R	-.029 (.001, .017)	-.066 - .007	
	RSA-R	.312 (.187, .790)	-.708 - 2.846	
3	PV x RSA-R	-1.368 (-.039, 1.464)	-3.613 - 1.327	.010
	PV x SCL-R	-.013 (-.001, .025)	-.047 - .036	
	SCL-R x RSA-R	-.013 (.168, .650)	-1.126 - 1.670	
4	PV x SCL-R x RSA-R	.757 (-.118, .942)	-.806 - 2.142	.006

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization, SCL-R = skin conductance reactivity, RSA-R = respiratory sinus arrhythmia reactivity.

Gender coded: -1 = Male, 1 = Female.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 12.

Regression Models for Relational Victimization, Gender, SCLR, and RSAR Predicting Anxiety Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% CI	ΔR^2
1	PV	.083 (.001, .038)	.013 - .166	.033**
2	RV	.039 (.000, .018)	.003 - .074	.053*
	Gender	.042 (.001, .027)	-.010 - .097	
	SCL-R	-.027 (.000, .014)	-.057 - .003	
	RSA-R	.267 (.023, .454)	-.668 - 1.312	
3	RV x Gender	-.038 (.000, .019)	-.077 - .001	.017
	RV x RSA-R	-.123 (-.089, .482)	-1.019 - .553	
	RV x SCL-R	.003 (.002, .013)	-.001 - .035	
	RSA-R x SCL-R	-.242 (.000, .376)	-1.001 - .564	
	RSA-R x Gender	.031 (.105, .620)	-1.176, 1.718	
	SCL-R x Gender	-.010 (.000, .017)	-.043, .022	
4	RV x Gender x RSA-R	-.332 (.006, .536)	-1.394 - .741	.012
	RV x Gender x SCL-R	.013 (.004, .016)	-.025 - .063	
	RV x SCL-R x RSA-R	.352 (.060, .415)	-.636 - 1.408	
	Gender x SCL-R x RSA-R	-.514 (-.234, .591)	-1.491 - -.110	
5	RV x Gender x RSA-R x SCL-R	-.414 (.150, .527)	-1.889 - 1.236	.003

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization, SCL-R = skin conductance reactivity, RSA-R = respiratory sinus arrhythmia reactivity.

Gender coded: -1 = Male, 1 = Female.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 13.

Regression Models for Relational Victimization, Gender, SCLR, and RSAR Predicting Depressive Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% CI	ΔR^2
1	PV	.131 (.005, .052)	.037 - .259	.053***
2	RV	.079 (-.001, .023)	.037 - .120	.065**
	Gender	-.008 (.002, .039)	-.089 - .075	
	SCL-R	-.029 (.000, .017)	-.064 - .005	
	RSA-R	.312 (.206, .811)	-.620 - 2.851	
3	RV x Gender	-.051 (.002, .032)	-.112 - .014	.050*
	RV x RSA-R	.004 (-.083, .710)	-1.268 - 1.096	
	RV x SCL-R	.009 (.000, .016)	-.022 - .040	
	RSA-R x SCL-R	.007 (-.071, .483)	-.856 - .745	
	RSA-R x Gender	-1.576 (.192, .991)	-4.252 - 1.019	
	SCL-R x Gender	-.020 (-.003, .023)	-.066 - .014	
4	RV x Gender x RSA-R	-1.095 (.000, .879)	-2.718 - .717	.024
	RV x Gender x SCL-R	.021 (.004, .028)	-.032 - .106	
	RV x SCL-R x RSA-R	.529 (-.076, .477)	-.312 - 1.223	
	Gender x SCL-R x RSA-R	-.610 (-.154, .836)	-2.173 - .519	
5	RV x Gender x RSA-R x SCL-R	-.338 (.313, .852)	-2.310 - 2.640	.001

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization, SCL-R = skin conductance reactivity, RSA-R = respiratory sinus arrhythmia reactivity.

Gender coded: -1 = Male, 1 = Female.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 14.

Regression Models for Physical Victimization, Gender, SCLR, and RSAR Predicting Anxiety Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% CI	ΔR^2
1	RV	.058 (.000, .015)	.028 - .088	.052***
2	PV	.068 (.002, .044)	-.013 - .167	.034 ⁺
	Gender	.042 (.000, .027)	-.009 - .096	
	SCL-R	-.027 (.000, .014)	-.057 - .003	
	RSA-R	.267 (.012, .445)	-.659 - 1.226	
3	PV x Gender	.011 (.001, .047)	-.088 - .105	.024
	PV x RSA-R	-1.298 (-.147, 1.034)	-3.049 - .247	
	PV x SCL-R	-.021 (.003, .021)	-.052 - .045	
	RSA-R x SCL-R	-.251 (.128, .397)	-1.009 - 1.158	
	RSA-R x Gender	-.179 (.082, .671)	-1.554 - 1.406	
	SCL-R x Gender	-.017 (.001, .016)	-.051 - .019	
4	PV x Gender x RSA-R	-.257 (-.186, 1.645)	-2.910 - 2.220	.019
	PV x Gender x SCL-R	.030 (.006, .036)	-.029 - .123	
	PV x SCL-R x RSA-R	.699 (.486, 1.025)	-1.505 - 5.297	
	Gender x SCL-R x RSA-R	-.610 (-.054, .699)	-2.184 - .571	
5	PV x Gender x RSA-R x SCL-R	-1.339 (.085, 1.477)	-5.872 - 1.720	.008

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization, SCL-R = skin conductance reactivity, RSA-R = respiratory sinus arrhythmia reactivity.

Gender coded: -1 = Male, 1 = Female.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

⁺ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 15.

Regression Models for Physical Victimization, Gender, SCLR, and RSAR Predicting Depressive Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% CI	ΔR^2
1	RV	.096 (.000, .022)	.054 - .141	.091***
2	PV	.080 (.004, .056)	-.017 - .205	.026
	Gender	-.008 (.000, .040)	-.087 - .071	
	SCL-R	-.029 (.001, .017)	-.064 - .004	
	RSA-R	.312 (.174, .796)	-.680 - 2.823	
3	PV x Gender	.085 (.002, .067)	-.060 - .220	.058*
	PV x RSA-R	-1.054 (-.007, 1.514)	-3.322 - 1.883	
	PV x SCL-R	-.014 (.000, .026)	-.054 - .041	
	RSA-R x SCL-R	-.047 (.095, .455)	-.853 - 1.211	
	RSA-R x Gender	-2.283 (.124, 1.094)	-4.980 - .130	
	SCL-R x Gender	-.032 (.000, .023)	-.081 - .017	
4	PV x Gender x RSA-R	-1.599 (.283, 2.393)	-5.679 - 3.796	.010
	PV x Gender x SCL-R	-.008 (-.002, .044)	-.088 - .070	
	PV x SCL-R x RSA-R	.406 (-.006, 1.124)	-1.807 - 2.568	
	Gender x SCL-R x RSA-R	-.228 (-.237, 1.005)	-2.271 - 1.004	
5	PV x Gender x RSA-R x SCL-R	.920 (.138, 2.070)	-3.708 - 5.142	.002

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. PV= physical victimization, RV= relational victimization, SCL-R = skin conductance reactivity, RSA-R = respiratory sinus arrhythmia reactivity.

Gender coded: -1 = Male, 1 = Female.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

Table 16.

Follow-Up Regression Models for Gender, SCL-R and RSA-R Predicting Anxiety Symptoms

Step	Variable	<i>b</i> (bias, SE)	95% CI	ΔR^2
1	Gender	.043 (.000, .027)	-.010 - .094	.024
	SCL-R	-.023 (.000, .014)	-.052 - .004	
	RSA-R	.494 (.071, .494)	-.296 - 1.849	
2	Gender x SCL-R	-.016 (.001, .015)	-.047 - .017	.010
	Gender x RSA-R	-.558 (.108, .587)	-1.797 - 1.051	
	SCL-R x RSA-R	-.176 (.088, .354)	-.754 - .930	
3	Gender x SCL-R x RSA-R	-.375 (-.151, .597)	-1.780 - .282	.003

Note: *bs* are unstandardized coefficients at the predictor's entry into the equation. . PV= physical victimization, RV= relational victimization.

Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Bold font indicates statistical significance at $p < .05$ for the 95% CI.

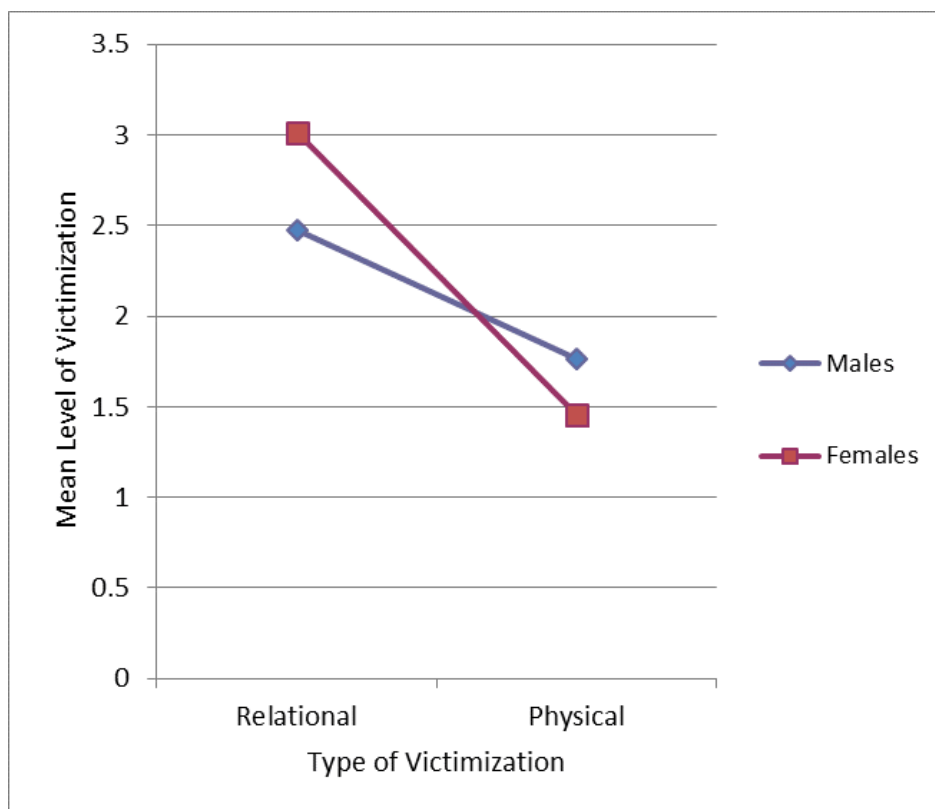


Figure 1. Gender differences in forms of victimization.

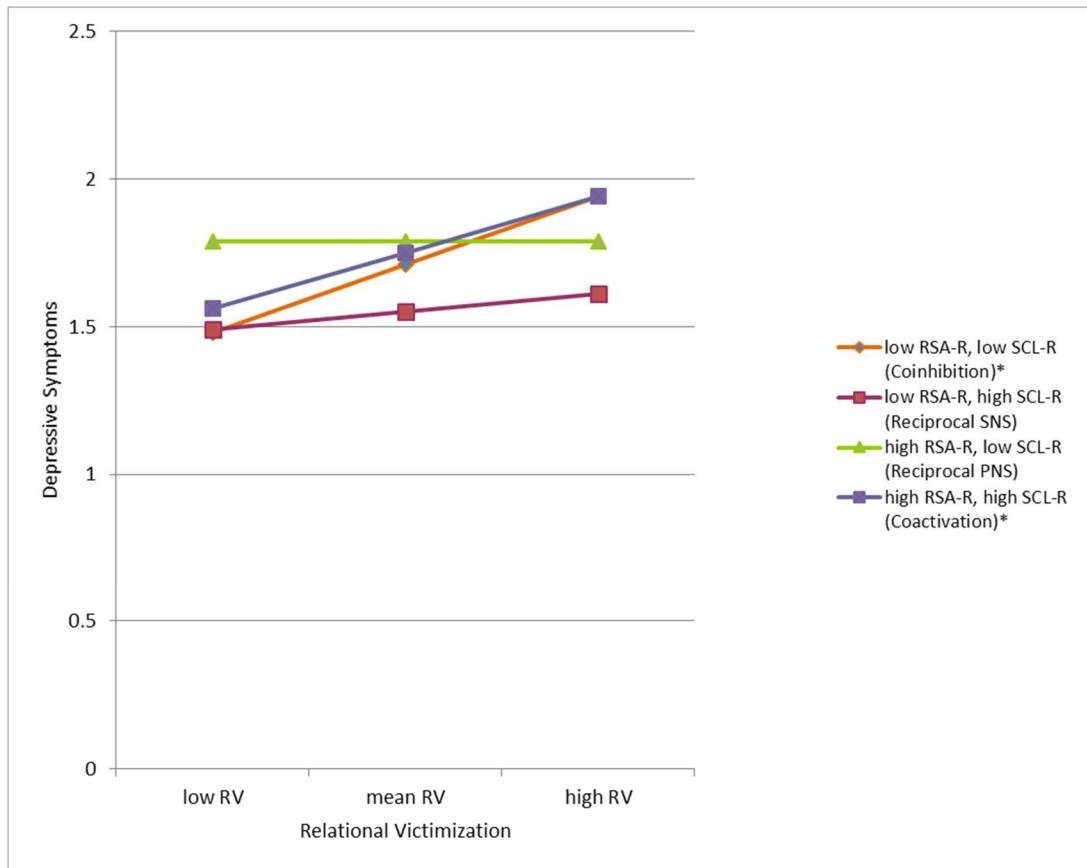


Figure 2. Three-way interaction between relational victimization, RSA-R, and SCL-R in the prediction of depressive symptoms. Low RSA-R indicates greater withdrawal while high RSA-R indicates augmentation.

* indicates statistical significance at $p < .05$.